ETIOLOGY OF LATE ONSET SEIZURES

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DECLARATION

I hereby declare that the dissertation title **"ETIOLOGY OF LATE ONSET SEIZURES"** was done by me at Stanley medical college and hospital during the year 2008-2009, under the guidance and supervision of Prof. MAGESH KUMAR, M.D. Professor of Therapeutics, Department of Internal Medicine.

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CERTIFICATE

This is to certify that this dissertation entitled **"ETIOLOGY OF LATE ONSET SEIZURES"** submitted by Dr.S.PRATIBHA to The Tamil Nadu Dr.M.G.R. Medical University Chennai is in partial fulfillment of the requirement of the award of M.D DEGREE BRANCH I (General medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of Professor & HOD **Signature of Dean**

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INTRODUCTION & LITERATURE REVIEW

DEFINITION

A 'seizure' (meaning "to take possession of") is a paroxysmal event due to abnormal, excessive, hyper synchronous discharges from an aggregate of central nervous system neurons. Depending on the distribution of discharges, this abnormal CNS activity's manifestations can range from dramatic convulsive activity to experiential phenomena unidentified by the observer[3]. A seizure needs to be distinguished from epilepsy. Epilepsy is described as the condition in which the patient has recurrent seizures due to a chronic, underlying process. This definition implies that a person with a single seizures, or recurrent seizure due to correctable or avoidable circumstances, does not necessarily have epilepsy. Epilepsy can be defined as 2 or more unprovoked seizures[3].

TYPES

Seizures are separable as convulsive and non convulsive, depending on the prominence of motor features. They are also distinguished as *focal* or *non focal*. Seizures with an immediate or proximate cause, such as an acute metabolic disturbance, infection, or head trauma, are *symptomatic* or *provoked*. In other instances, seizures

that may result from past brain injury can be described as *remote symptomatic*.

In large number of people, a cause is not identifiable. These seizures are classified to be either *idiopathic* or *cryptogenic.* The term idiopathic refers to disease from an unknown cause and is presumed to have a genetic basis. The term cryptogenic implies a symptomatic cause undiagnosable with current medical technology[2].

CLASSIFICATION OF SEIZURES:

International league against Epilepsy Classification of epileptic seizures[2]:

I. Partial(focal, local) seizures

A. Simple partial seizures (consciousness not impaired)

1. With motor symptoms
2. With somato sensory symptoms
3. With autonomic symptoms
4. With psychic symptoms

B. Complex partial seizures (with impairment of consciousness)

1. Beginning as simple partial and progressing to loss of consciousness
2. With no other features
3. With features as in simple partial seizures
4. With automatisms

C. With impairment of consciousness at onset

1. With no other features
2. With features as in simple partial seizures
3. With automatisms

D. Partial seizures evolving to secondarily generalised seizures

1. Simple partial seizures evolving to generalised seizures.
2. Complex partial seizures evolving into generalised seizures

3. Simple partial seizures evolving to complex partial and then to generalised seizures.

II. Generalised seizures (convulsive or non convulsive)

A. Absence seizures (typical/ atypical)

B. Myoclonic seizures

C. Clonic seizures

D. Tonic seizures

E. Tonic-clonic seizures

F. Atonic (astatic) seizures

III. Unclassified epileptic seizures

Includes all seizures that cannot be classified because of inadequate or incomplete data and some that defy classification hitherto described categories.

PARTIAL SEIZURES

These occur within the discrete regions of the brain. Simple partial seizures and complex partial seizures are distinguished solely on

the basis of consciousness. Consciousness is usually assessed by the ability to respond to external stimuli.

This ability is intact in SPS but impaired in CPS.

SIMPLE PARTIAL SEIZURES

This is further divided according to symptoms as motor, sensory, autonomic and psychic. Motor sensations can vary and include motor signs with or without march, versive movements, posturing and phonatory symptoms.SPS with sensory symptoms includes all 5 senses plus a vertiginous sensation. SPS with automatic symptoms include the common rising epigastric sensation typically seen in mesial temporal lobe epilepsy(MTLE)and less frequent symptoms such as vasomotor phenomena or mydriasis. SPS with psychic symptoms are characterised by various experiences involving memory (eg., déjà-vu, jamaisvu), affect (fear, pleasure), or other complex, psychic phenomenon such as illusions.

COMPLEX PARTIAL SEIZURES

CPS includes complex symptomatology like automatisms and impairment of consciousness. The seizures consists of involuntary but coordinated motor activity that is purposeless and repetitive. Common

automatisms include lip smacking, chewing, fidgeting and walking. CPS can begin as SPS and seizures with impaired consciousness. Electroencephalographically, partial seizures are characterised by focal epileptic form discharges interictally (spikes or sharp waves) and ictally.

GENERALISED

ABSENCE SEIZURES

These are characterised by sudden brief lapses of consciousness without loss of postural control. Absence seizures constitute 15-20% of childhood seizures. In atypical absence, there is less abrupt onset, and termination with longer duration. EEG typically shows generalised symmetric 3 Hz spike and wave discharge that begins and ends suddenly on a normal EEG background.

MYOCLONIC SEIZURES

It is a sudden and brief muscle contraction that may involve one part of the body or the entire body. EEG shows bilateral synchronous spike and wave discharges.

CLONIC SEIZURES

These are repetitive rhythmic clonic movements that are bilateral

and symmetric and associated with fast activity or spike-wave complexes on EEG, which evolve over time typically decreasing in frequency while increasing in amplitude.

TONIC SEIZURES

These are characterised by stiffening of the musculature mostly axial, but also appendicular associated with low voltage paroxysmal fast activity.

GENERALISED TONIC-CLONIC SEIZURES (GTCS)

This is the sequential combination of previous two types in which the tonic phase is gradually interrupted by quiescence evolving to the clonic phase with rhythmic spikes and spike-wave complexes decreasing in frequency on EEG.

ATONIC SEIZURES

Characterised by sudden loss of postural muscle tone lasting for 1-2 secs. Consciousness is briefly impaired but usually there is no post ictal confusion. EEG shows generalised epileptiform discharges (spikes, spike-wave complexes) or an abrupt flattening on EEG.

INCIDENCE AND PREVALENCE

Studies have estimated that 1.5% to 5.0% of persons in any population will have a seizure at some time of their lives[2].Incidence of epilepsy is 0.3-0.5 % in different populations throughout the world and prevalence has been estimated to at 5-10 persons per 1000[3].

In developed countries, the incidence rates range from 40 to 70 per 100000. But in developing countries, the rates may be as high as 100 to 190 per 100000[2]. Partial seizures, with or without secondary

generalisation, are the most common seizures, followed by generalised tonic- clonic seizures. Factors that contribute to higher prevalence of epilepsy in developing countries include limited access to health care which compounds the problems of birth injury and head trauma. Poor sanitation leads to high incidence of CNS infections which cause seizures. Poverty and illiteracy increases the risk of social diseases like alcoholism and substance abuse which can contribute to development of seizures. Further influencing the treatment of seizures is combination of local social perceptions, government policies and anti-epileptic drug availability (Burneo et al., 2005[5]; de Bittencourt et al., 1996[6]; Preux and Dreuet-Cabanac, 2005[7]).

Harrison[3] mentions following as the causes of seizures in ages > 40 yrs:

- Cerebrovascular disease
- Brain tumour
- Alcohol withdrawal
- Metabolic disorders (uremia, hepatic failure, electrolyte abnormalities, hypoglycaemia)
- Alzheimers disease and other degenerative disorders

- *Idiopathic*

The term 'incidence-prevalence gap' refers to higher incidence of epilepsy in developing countries than in developed countries, whereas prevalence values are similar throughout the world (Bharucha NE et al, 2003[8]). Possible explanations are differences in methodology, namely, inclusion of acute symptomatic seizures as incidence in developing countries: higher mortality rates in developing countries; the higher rates of remission, which would imply more benign prognosis. There are no answers yet to these speculations, nor will be until there are well-conducted incidence studies and population based outcome studies from developing countries.

The incidence of epilepsy is bimodal in developed countries (Forsgren et al., 2005[9]). Rates are high in the first decade, particularly before the age of 1 year, and decline during childhood, reaching a minimum between 20 and 30 years of age. A secondary rise in the incidence occurs after 60 years, increasing dramatically with age (Brodie and Kwan, 2005[10]).

This bimodal distribution is not as evident in developing countries. The age-specific incidence remains high throughout

adulthood, largely related to symptomatic seizures occurring as a result of infection and trauma (de Bittencourt et al., 1996[6]).

Men are 1.0 to 2.4 times more likely to have seizures than women[2].

CAUSES OF SEIZURES IN ADULTS:

The cause of great majority of seizures and epilepsy in adult life will be symptomatic. But in studies of hospital- and clinic-based populations, as well as in field studies, the etiology of epilepsy is identifiable in only one fourth to one third of the cases [2].

I. **ACUTE SYMPTOMATIC SEIZURES**

These seizures, otherwise called reactive seizures/ provoked seizures/ situation related seizures. These occur at the time of a systemic insult or in close temporal association with a documented brain insult. These seizures need only a short acting benzodiazepine as treatment and do not require a long term treatment.

Acute symptomatic seizures typically occur within first week of an insult known to predispose to seizures (eg, head trauma, CNS infection, cerebrovascular disease, brain tumour, metabolic causes, drug

withdrawal). These seizures predispose later to unprovoked seizures, the incidence of it depending upon the initial cause and severity of the event[1].

1. DISORDERS OF FLUID AND ELECTROLYE IMBALANCE

a. SODIUM ELECTROLYTE DISORDERS

Hyponatremia is commoner than hypernatremia and is seen commonly in congestive cardiac failure, liver disease, nephrotic syndrome. The rapid change in osmotic gradient is compensated by movement of water molecules into cells leading to cerebral edema and hence seizures [2]. Mostly seizures are generalised tonic- clonic in nature, convulsions are noted in 9% of patients with sodium concentrations<125 mmol/L. Typical neurologic manifestations of acute symptomatic hyponatremia consist of generalised tonic – clonic seizures, hypothermia and respiratory failure.

In diuretic induced acute hyponatremia, seizures are usually generalised tonic clonic, with frequent progression to status epilepticus. (Johnston C, et al, 1989[11]).

In SIADH neurological manifestations are disturbances of

consciousness and especially generalised tonic clonic seizures. Persistent simple partial motor seizures and multi focal seizures have been reported in SIADH, acute intermittent porphyria. Mortality has been reported in 50% of adult patients especially women and 8.4% of children. (Arieff AI et al, 1985[12]).

Hypernatremia can occur in fever, gastroenteritis, burns and diabetes or be due to gross fluid restriction. Seizures are most commonly seen in patients who are uremic and acidotic.

b. CALCIUM ELECTROLYTE DISORDERS

Hypocalcemia may be seen in hypoparathyroidism, vitamin D deficiency, acute pancreatitis, pseudohypoparathyroidism. Up to 70% of patients with hypoparathyroidism may have seizures associated with tetany and altered consciousness. Hypercalcemia commonly occurs in disseminated malignant disease, like in bronchogenic carcinoma. It occasionally can lead to generalised seizures.

c. HYPOMAGNESEMIA

Direct co relation between low plasma magnesium concentrations, seizure frequency and status epilepticus has been demonstrated in generalised idiopathic seizures. It is suggested that

hypomagnesemia may remove inhibitory influence from NMDA glutamate receptors. Then, this process may trigger neuronal depolarisation. Magnesium specially inhibits sodium flux through NMDA type glutamate receptors.(Dharnidharka VR et al, 2005[13]).

Hypomagnesemia may be found in inflammatory bowel disease, bowel resection, other malabsorption syndromes, after removal of parathyroid neoplasm, diabetic acidosis, excessive use of diuretics, septicaemia and closed heart surgery.

Generalised and partial seizures have also been described in patients with hypomagnesemia induced by the ketogenic diet for intractable epilepsy, cyclosporin A therapy and ibuprofen overdose.

Hypomagnesemia also associated with carpopedal spasm, neuromuscular irritability resulting in action tremor, myoclonic jerks, tendon hyperreflexia.

2. METABOLIC DISORDERS:

a. DISORDERS OF GLUCOSE METABOLISM:

Seizures are particularly common in association with non ketotic hyperosmolar coma. Focal motor seizures appear particularly common.

Neurological manifestations such as seizures, focal neurologic signs, myoclonic twitches, nystagmus and meningeal signs often provide the first clinical clues to the presence of nonketotic hyperglycemia. Extra cellular glucose excess has proconvulsant activity even without previous focal lesion. Hyperglycemia may precipitate seizures by lowering gamma – aminobutyric acid levels, resulting in lower seizures threshold. (Schwechter EM et al, 2003[14]).

Seizures occur in about 25% of patients and are the chief initial manifestation in 6%. Seizures are most often partial motor seizures, occurring in 75% to 86% of cases. The seizures are frequent and repetitive and are often followed by transient postictal paralysis. Typical jacksonian seizures have also been reported. Generalised tonic - clonic seizures have been described less frequently(Grant C, et al, 1985[15]).

Such seizures may be very resistant to antiepileptic treatment but respond rapidly to the correction of hyperglycemia. Seizures are rare in ketacidotic coma. (Sabharwal RK et al, 1989[16]).

Hypoglycaemia is usually seen in diabetic patients using insulin or oral hypoglycaemic drugs. Rarely it can occur in patients with gi endocrine tumours like insulinoma, in severe liver disease or in

alcoholic with poor glycogen reserve, sepsis, fasting, gastroenteritis with diarrhea and dehydration, adrenocortical insufficiency. Confusion, bizarre behaviour, obtundation, stupor, or for coma occur in more severe degrees of hypoglycemia. Hypoglycaemic seizures occur in 10% to 20% of adults and more frequently in children, (Lancet, 1985[17]). Usually seizures are generalized tonic and tonic- clonic in type. Partial seizures typically occur in adults. Insulin- induced hypoglycaemic convulsions could be mediated by serotoninergic, dopaminergic, and excitatory amino acid pathways. (Anuradha K et al, 2004[18])

b. **THYROID DISORDERS**

HYPOTHYROIDISM

Seizures are more common in hypothyroidism than in hyperthyroid state. It s seen in myxedema coma or may be due to underlying hyponatremia. Generalized tonic – clonic seizures and EEG findings of low – voltage slow waves and dysrhythmic background suggest a nonspecific encephalopathy. Seizures may be characterized by an unexplained prolonged postictal recovery. (Bryce GM et al, 1992[19]). Patients are not at continued risk after the underlying abnormality is corrected. Treatment with excess thyroxine can cause seizures. This

thyroxine induced epilepsy with hyper motor seizures is controlled by reducing thyroxine doses.

HYPERTHYROIDISM

Both generalized tonic – clonic and partial motor epileptic seizures associated with or followed by signs of hyperthyroidism, irritability, tachycardia, tremor, anxiety, confusional state have been reported as presenting symptoms of thyrotoxicosis.(Jabbary B et al, 1980[20]).

HASHIMOTO'S ENCEPHALOPATHY

In this disorder, frequently epileptic seizures are presenting symptom. These include generalized and partial seizures as distinguishing features. These seizures are resistant to antiepileptic medications but successfully treated with corticosteroids. (Mckeon A, et al, 2004[21]).

c. PORPHYRIA

Seizures may occur in 15% of patients with acute intermittent porphyria. Control of seizures can be significant management problem as phenytoin, phenobarbitone and carbamazepine can all induce attacks of porphyria.

d. LIVER DISEASE

Seizures are a feature of acute hepatic failure and in patients with high grades of hepatic encephalopathy. They may be focal but more commonly generalised tonic clonic. Seizures are frequent neurologic complication of liver transplantation, affecting 10% to 29% of cases. Single or recurrent generalized tonic – clonic seizures are the most common ictal manifestation. (Lopez OL et al, 1992[22])

e. RENAL FAILURE

In renal failure, seizures may be caused due to uremic encephalopathy, aluminium encephalopathy in infancy and childhood, dialysis disequilibrium syndrome and dialysis encephalopathy syndrome. Acute uremic encephalopathy causes convulsions in as many as one third of the patients. Most are generalised tonic clonic seizures. Uremia is characterized by retention of toxic metabolites derived mainly from proteins associated with changes in volume and electrolyte composition of the body fluids and excess of deficiency of various hormones. Renal failure results in a gradual accumulation of several substances, but no single metabolite has been identified as the sole cause of uremic encephalopathy (Bergstrom J et al, 1985[23]). Seizures can also

occur during dialysis (dialysis equilibrium syndrome) and as a part of dialysis encephalopathy. Dose reduction of anti epileptic drugs like phenytoin and carbamazepine is necessary.

f. DRUG RELATED SEIZURES

Drugs and particularly alcohol are common cause of seizures. Most commonly involved drugs are penicillin, hypoglycaemic drugs, lignocaine and psychotrophic drugs. Most drug induced seizures are dose related. Patients with renal failure and hepatic failure are particularly prone to seizures because of inability to effectively metabolise and excrete the drugs.

Withdrawal of chronically administered sedative drugs which show tolerance is a well recognised cause of seizures and may occur with alcohol, barbiturates and benzodiazepines. The best studied seizures are those with alcohol abuse or withdrawal, sometimes as a part of delirium tremens. Such an abuse is an important cause of seizures in the community. It must be considered in adults developing tonic- clonic seizures for the first time.

Seizures associated with alcohol may be a consequence of acute or remote head injury, CNS infection, stroke, or metabolic derangement

(like hypoglycaemia). But in many heavy drinkers, seizures are usually a direct effect of the ethanol, especially as a withdrawal phenomenon. The risk is clearly related to the dose of alcohol consumed. The abrupt, absolute or relative withdrawal is most commonly responsible for causing seizures. If beyond withdrawal period (7 to 30 hrs after the last drink), or with focal signs, underlying structural pathology has to be ruled out[1].

II. REMOTE SYMPTOMATIC SEIZURES

1. HEAD INJURY/ POST- SURGICAL:

Head injury increases the risk of unprovoked seizures, with the greatest risk occurring in survivors with severe injury. Severe injuries with more than 24 hours of unconsciousness or post traumatic amnesia, brain contusion or intracerebral hematoma increased the risk of seizures by 29- fold (Rocca WA et al, 1987[24]).

Analogous to seizures after penetrating injuries, unprovoked seizures may be a consequence of neurosurgical procedures to the head. Studies for unprovoked seizures after neurosurgical procedure are complicated by the nature of the underlying disorder, the presence of seizures before surgery, and the select nature of the population studied.

Foy PM et al, 1981[25] in their study on natural history of post operative seizures on 877 consecutive neurosurgical patients, unprovoked seizures developed in 17% of patients within 5 years.

2. CNS INFECTIONS:

CNS infection increased the seizure risk by 11-fold. A 16-fold risk was associated with viral encephalitis, a 4-fold risk with bacterial meningitis, a 2-fold risk with aseptic meningitis. Most of the unprovoked seizures occurred within the first 5 years of infection (Annegers JF et al, 1988[26]).

PYOGENIC MENINGITIS:

Pyogenic bacteria induce formation of a purulent exudate within the subarachnoid space related to migration of neutrophils and other immune cells.

Such exudates, together with direct effects of bacterial toxins may cause seizures by; (1) occlusion of small pial arteries, (2) venous thrombosis, (3) diffuse brain swelling,(4) toxic effects of bacteria in subpial space, (5) acute metabolic changes[2].

Acute symptomatic seizures occur in 40% of patients, more often

generalised tonic clonic seizures. 2% to 7% of the patients developed chronic epilepsy, mostly in those with permanent neurological deficits (Pomeroy SL et al, 1990[27]).

VIRAL ENCEPHALITIS:

HSV-1 is the most common cause of sporadic viral encephalitis.

Viruses cause diffuse swelling, vascular congestion, demyelination, inflammatory infiltrates, microglial proliferation, diffuse necrosis of cerebral cortex and basal ganglia. Seizures are mostly related to development of cerebral infarcts. Seizures may be generalised or partial, more often recurrent and persist after the acute disease (Kennedy PGE et al, 2004[28]).

CEREBRAL MALARIA:

Malaria is endemic in our country. Acute cerebral malarial encephalopathy still has 22% mortality inspite of improved treatment[2]. Patient usually presents with unarousable coma, evidence of P.falciparum infection and no other identifiable cause of coma. There is extravasation of erythrocytes resulting from endothelial damage causing cytokine release in patients with cerebral malaria. There is also plugging

of capillaries by parasitized erythrocytes. This causes brain damage as a result of obstruction to cerebral vasculature and leads to ischemic hypoxia. Seizures occur in about 70% of the cases, and are most often generalised tonic clonic, although some patients may present with partial seizures (Roman GC et al, 1992[29])

NEUROCYSTICERCOSIS:

Neurocysticercosis is caused by the larvae of the tapeworm *Taenia solium* in the nervous system, a disease suffered by millions of people living in the developing countries. In these areas, the disease accounts for up to 12% of all admissions to neurological hospitals and is the leading cause of acquired epilepsy in adults. More than 50,000 new cases of NCC-related deaths occur annually, and of the many more patients who suffer related neurological sequelae, most are affected at productive age. This makes NCC a large public health problem in developing countries.

Stages of inflammation through which cysticerci pass through are colloidal, granular, calcified. Inflammatory reactions around cysticerci induce pathological changes in CNS serving as a substrate for further development of seizures. It causes granular ependymitis which leads to

obstructive hydrocephalus. Seizures, focal deficits, cognitive decline and increased intra cranial pressure are common manifestations of NCC. Seizures occur in about 70% of the cases (Del Brutto OH et al, 1992[30], Rajshekhar V et al, 2004[31]). NCC is often diagnosed on the basis of information provide by neuroimaging studies and serology. Imaging studies give information about the number and location of lesions as well as on the stage of evolution of cysticerci.

CNS TUBERCULOSIS:

Tuberculous meningitis is a sub acute disease characterised by fever, malaise, behavioural changes, headache, seizures, focal neurological signs, and stupor or coma. Seizures occur in approximately 20% of the patients, are more common in children than in adults, and may represent predictors of poor outcome (Hosoglu S et al, 2002[32]). Intracranial tuberculomas present as mass lesions with increased intra cranial pressure, focal signs and seizures (Garcia- Monco JC et al, 2005[33]).

BRAIN TUMOURS:

Although neoplasms of the brain account for only 1% of cases of epilepsy, seizures occur in approximately 50% of children with supratentorial tumours and seizures develop in approximately 35% to 40% of adults with brain tumours. (LeBlanc FE et al, 1974[34]). The rate is much lower for tumours of the infratentorial or pituitary region, and consequently even higher for supratentorial lesions. Seizures occur especially commonly in association with oligodendrogliomas. Seizures are also commonly encountered in patients with meningiomas. In contrast, the incidence of seizures in patients with cerebral metastasis is much lower; most reports suggest an incidence of 20% at time of presentation. (Gamache FW, et al, 1979[35]). Imaging increases the probability of finding an early malignancy. So it is recommended that every patient who presents with seizures for the first time undergoes brain imaging[2]. EEG is also useful in assessing these patients. Findings correlate with tumour location, and in approximately 40% of the patients, the EEG abnormalities are lateralised to the side of the tumour. EEG is relatively inexpensive and helps to localise the epileptogenic foci by a method different from neuro imaging. Patients with brain tumours who undergo surgeries have increased risk of unprovoked

seizures. 12% to 16.3% of newly diagnosed epilepsy have brain tumours, indeed, seizures are often the first sign of brain tumour (Ludhorf K et al, 1986).

CEREBROVASCULAR DISEASE:

A chronic epileptogenic lesion at the stroke site may account for unprovoked seizures that occur more than 1 to 2 weeks after a clinical stroke. Such unprovoked seizures occur after a clinically detected stroke in 2.7% to 35% of patients*1. This variation in incidence may due to difference in the methodology of studies done. Regardless of the selection factors or length of follow – up, the risk for unprovoked seizure after stroke is at least three times (Hauser WA et al, Minnesota, 1935- 1984[36]).

Because stroke produces a focal brain injury that serves as a substrate focus for seizure development, it has been hypothesized that only unprovoked partial seizures should occur after stroke. Generalized seizures are, however, not uncommon after stroke, although some investigators include secondary generalized seizures in this category. Researchers who distinguish primary from secondary generalized seizures find that primary generalized seizures account for 4% to 69% of

all unprovoked seizures after stroke. Undetected onset of partial seizures may account for this variability. Nonetheless, true primary generalized seizures do occur after stroke, perhaps as a result of persistent global alterations in neurotransmitter function after stroke or of factors that alter cerebral autoregulation in people with risk factors for stroke.

Cleary P et al, Lancet2004[37], in their studies state that the cumulative risk for stroke in patients with seizures was 10.0%, compared to 4.4% in patients without seizures. This increased prevalence of seizures in patients with stroke predicted that risk factors for stroke may also increase the risk factors for seizures. Studies state that hypertension, left ventricular hypertrophy independently increase the risk of unprovoked seizures (Herdorffer DC et al, 1996[38]).

Cortical venous stroke with underlying ischemia and infarction is highly epileptogenic[4]. The same is true for hypertensive encephalopathy and Thrombotic Thrombocytopenic Purpura (TTP), which has strong tendency to cause non-convulsive status epilepticus. The rupture of saccular aneurysm may be marked by one or two generalised convulsions. Subcortical cerebral haemorrhages occasionally become a source of recurrent focal seizures[4].

HYPOXIC-ISCHEMIC ENCEPHALOPATHY:

Cardiac arrest, suffocation or respiratory failure, carbon monoxide poisoning and other causes of hypoxic encephalopathy tend to induce diffuse myoclonic jerking and generalised seizures as cardiac function is resumed. It may persist indefinitely as intention myoclonus-convulsive state (Lance-Adams syndrome)[4].

DEMENTIA:

The underlying pathology of Alzheimers may be associated with increased susceptibility of seizures, with increase by 10- folds (Hauser WA et al, 1986[39]). All the seizures are usually generalised tonic clonic seizures (Romanelli MF et al, 1990[40]).

Hesdorffer et al, 1996[41] evaluated the risk for seizures associated with dementia and found that dementia increased the risk of seizures by 8- fold.

EEG IN SEIZURES:

EEG provides confirmation of Hughling Jackson's concept of epilepsy- that it represents a recurrent, sudden, excessive discharge of cortical neurons. EEG is undoubtedly the most sensitive, indeed

indispensable tool for diagnosis of epilepsy, but like other ancillary tests, it must be used in conjunction with clinical data. It helps in confirming the diagnosis, and sometimes in localisation of the focus. The region of earliest spike activity corresponds best to the epileptogenic focus. This rule guides in epileptogenic surgeries. The post-seizure or post-ictal state following generalised seizures also has its EEG correlate, taking in the form of random generalised slow waves. Following partial or focal seizures, EEG shows focal slowing. With clinical recovery, EEG returns to normal or to pre- seizure state. A single EEG tracing obtained during inter ictal state is abnormal in 30 to50 % of the patients; this figure rises to 60 to70 %if the patients are subjected to three or more studies using standard activating measures (hyperventilation, photic stimulation, sleep). With structural lesions, focal slow and sharp activity, which is not clearly epileptiform, may be the only clue to a seizure focus.

Following are few similar studies, which evaluate late onset seizures:

1. Acta Neurol Scand. 1982 Aug;66(2):216-26

Late onset epilepsy. A prospective study.

Ahuja GK, Mohanta A[46].

253 cases of late onset epilepsy were studied prospectively. 27 cases (10.7%) had space-occupying lesion, 19 cases (7.5%) had cerebrovascular disease, 13 cases (5.1%) cerebral cysticercosis and 4 cases (1.6%) had diffuse cerebral atrophy. No cause could be detected in 190 cases (75.1%). Analysis of clinical data and radiological studies showed that a majority (85%) of patients with 'tumour' who presented with epilepsy had focal neurological deficit and/or papilloedema. Focal slow-wave abnormality in EEG also gave an indication of an organic lesion. Patients who had epilepsy for more than 1 year, infrequent attacks and partial complex seizures, were less likely to have a tumour.

2. *Epilepsia* 2000;41 Suppl 9:31-35

Epilepsy in the Elderly.

Hiyoshi T, Yagi K[47].

Epilepsies were classified as generalized in 33 patients (17.4%),

partial in 145 (76.3%), and undetermined in 12 (6.3%). Twenty-nine of 33 patients with generalized epilepsy were idiopathic, whereas all patients with partial epilepsy were symptomatic. Patients with late onset (50 years or older) had no family history of epilepsy, and half of them had a past history of cerebrovascular disease or head injury as a presumed etiology. In patients with idiopathic generalized epilepsy (IGE), 25 of 29 had early onset, and a family history of epilepsy was found in 31%. Nineteen patients continued to have seizures after 50 years of age, albeit infrequently. Furthermore, 10 of them showed exacerbation around the age of 50 yrs.

3. Age Ageing. 1982 Feb;11(1):24-8

Epileptic seizures in the elderly: I. Aetiology and type of seizure. Roberts MA, Godfrey JW, Caird FI[48].

Eighty-one elderly patients with epileptic seizures are described, of whom 60 were investigated by computed tomography. The cause was cerebrovascular disease in 44%, tumour in 12%, extracerebral in 11%, and unknown in 16%. Partial seizures were commoner in patients with tumour than with other causes.

4. Epilepsia. 2001 Dec;42(12):1594-9

Prospective study of seizures in the elderly in the Marshfield Epidemiologic Study Area (MESA).

Ruggles KH, Haessly SM, Berg RL[49].

Forty-eight patients having a first seizure were identified (162 of 100,000). Twelve patients had recurrent seizures, and 36 had a single seizure at the time of study entry. Fourteen of these 36 had had an abnormal MRI, CT, or EEG. The remaining 22 had a single seizure and normal imaging and EEG. Six of these had one or more subsequent seizures, and all six were in the group with normal tests. Etiologies included vascular, neoplasm, trauma, dementia, metabolic, and unknown. Seventy-five percent of the patients achieved seizure control with phenytoin, carbamazepine, and/or valproate. Twenty-seven percent experienced adverse side effects.

5. Schweiz Med Wochenschr. 1990 May 26;120(21):787-92.

Initial epileptic crisis after the age of 60: etiology, clinical aspects and EEG

Henny C, Despland PA, Regli F[50].

Retrospectively a series of 100 in patients with onset of epileptic

seizure after the age of 60 were reviewed. All of them were investigated by EEG and 96 by CT scan. The most frequent cause of seizure was previous stroke, with 25 cerebral infarcts and 5 hemorrhages. Neoplastic lesions were present in 18 cases, with glioma (high grade), meningioma and metastases in the same proportion. Other etiologies included toxico-metabolic (18 cases), post-traumatic (9 cases), cerebral atrophy (4 cases) and miscellaneous (14 cases). The causes of seizure remained unknown in 7 patients, of whom 6 had focal signs in either clinical examination or EEG. Focal seizures (with or without secondary generalization) accounted for 65% of all cases and generalized seizures for 35%. The EEG was normal in 12 patients and abnormal in 88, with diffuse slowing in 55 patients and focal signs in 70 (some patients had both diffuse slowing and focal signs).

6. **Postinfarction seizures. A clinical study**

SR Gupta, MH Naheedy, D Elias and FA Rubino [51]

Department of Neurology, Veterans Administration Hospital, Hines, Illinois[51]

We retrospectively studied 90 patients with postinfarction seizures to determine the clinical features (onset, number, type), prognosis, and electroencephalographic and computed tomographic findings; we included infarctions of all etiologies. Thirty-three percent

of the 90 seizures appeared early (within 2 weeks after the infarction), and 90% of the 30 early seizures appeared within 24 hours after the infarction. Seventy- three percent of the 90 seizures occurred within the first year, and only 2% occurred greater than 2 years after the infarction. Fifty-six percent of the 90 seizures were single, and status epilepticus was seen in only 8%. Early-onset seizures were more likely to be partial (57% of 30); late-onset seizures were more likely to be generalized (65% of 60). Thirty-nine percent of the 90 initial seizures recurred, and there was no significant difference in recurrence rate between early- or late-onset initial seizures. Twenty-two percent of the 90 initial seizures became multiple recurrent seizures, and we could identify a precipitating factor in 86% of the 35 recurrent seizures. The most common electroencephalographic abnormality in the 61 patients so examined was focal slowing (61%), but recurrent seizures occurred in 100% of the four patients with periodic lateralized epileptiform discharges and in 75% of the eight patients with diffuse slowing. Computed tomography in 61 patients showed that large infarctions were associated with early ($p < 0.021$) and multiple ($p < 0.05$) seizures. Deep infarctions on computed tomograms (cortical infarctions extending to sub cortical structures) tended to cause recurrent seizures ($p < 0.057$).

7. Epilepsia 26 (3): 227-231 1985

Late-Onset Epilepsy: Etiologies, Types of Seizure, and Value of Clinical Investigation, EEG, and Computerized Tomography Scan

Agnete Mouritzen , Fuglsang-Frederiksen , Ulla Svarre-Olsen Mogens[43]

Approximately 25% of patients with epilepsy will have their first seizure after the age of 25 years. These individuals will need special attention with regard to etiology. Brain tumor is one of several causes that may be suspected. The present study of 221 patients with late-onset epilepsy from the University Clinic of Neurology, Hvidorvre hospital, Copenhagen, Denmark, was undertaken to took for menas to select those cases in which computerized tomography (CT) scan should be performed. Brain tumor was the cause in 16% and cerebrovascular infarctions in 14%. The major etiological group was the one in which no cause could be detected (38%). Alcohol abuse as the etiology—defined as cases with a history of long-standing alcohol overuse, concomitant signs of alcohol intoxication, and spontaneous recurrent epileptic seizures—made up a group of one-fourth of all the patients with late-onset epilepsy. Comparison of the history, clinical symptoms and sings, EEG abnormalities, and CT scan speaks in favor of some consideration being given to the first three parameters before the CT scan is

performed.

AIM OF THE STUDY

- To study the clinical profile of patients with new onset seizures after 40 yrs of age

- To find the etiology of seizures in adult population using clinical methods, CT Scan and Electroencephalography

MATERIALS AND METHODS

50 patients, above 40 yrs of age, admitted with new onset seizures in medical wards of Government Stanley Hospital during June 2009 to October 2009.

Patients with history of seizures in childhood or any time in past were excluded from the study.

Patients with episodes mimicking seizures like syncope, TIAs and pseudo seizures were identified and excluded from the study.

A detailed history of presenting complaints, details of the seizures, relevant past and personal history were obtained from the patient and reliable attendees. Details of the seizures included:

- First attack
- Last attack
- Type of seizures
- Part of body involved
- Duration

- Frequency
- Maximum period of freedom
- Time of attack
- Precipitating factors
- Aura
- Post-ictal symptoms
- Automatisms

Past history included

- Hypertension, inc. PIH
- Diabetes mellitius
- Head injury
- Previous stroke
- Cardiovascular (RHD/ MVP/ IHD)
- Respiratory illnesses(COPD/ PTB/ BA)

- Renal transplant
- Psychiatric illnesses
- Jaundice

Personal history included

- Smoking
- Drugs
- Alcohol
- Snuff
- Other addictions
- Pork
- Sleep
- Sexual promiscuity

A detailed general examination, including vital signs recording, complete neurological examination and examination of other systems

was done.

Neurological examination included

- Neurocutaneous markers
- Focal deficits
- Confusional state
- Fundus

Investigations like Blood counts, Random blood sugar, Renal function tests, Electrolytes, Chest X Ray, ECG, CT Brain- Plain, and EEG were done for all patients.

CT Scans were noted for any clearly abnormal focal lesions. If suspicious lesions were found, a contrast CT Scan/ MRI was obtained depending on the situations.

In case of acute febrile illness or signs of meningeal irritation associated with the seizures,

- QBC- for malarial parasite and
- Cerebrospinal fluid analysis was done.

Lumbar puncture for CSF analysis was done under aseptic precautions. CSF was analysed for:

- Biochemistry: Sugar, proteins
- Cell count: Total, differential
- Cytology
- Culture and sensitivity
- Ziehl-nielson staining
- When these showed abnormalities, anti HSV antibodies were done when relevant.

In suspicious patients, like those who had tuberculoma-brain, viral encephalitis, HIV-ELISA and VDRL were done.

Cardiac evaluation was done for patients with abnormal physical findings in cardiovascular system, abnormal ECG and for all patients with cerebrovascular etiology (cerebral infarction and intracerebral haemorrhage).

EEG was done for all the patients irrespective of whether an

underlying cause was found or not.

The clinical profile, brain imaging findings, EEG findings were all analysed to derive and etiological diagnosis for the seizures

OBSERVATIONS

Table.1 AGE CHART

Age	GTCS	PSG	CPS	SPS	Total
40- 50 yrs	12	1	3	1	18 (36%)
50- 60 yrs	10	5	4	1	19 (38%)
>60 yrs	4	7	2	-	13 (!4%)
Total	26	13	9	2	50

Table.2 SEX CHART

Sex	Incidence
Male	31(62%)
Female	19 (38%)
Total	50

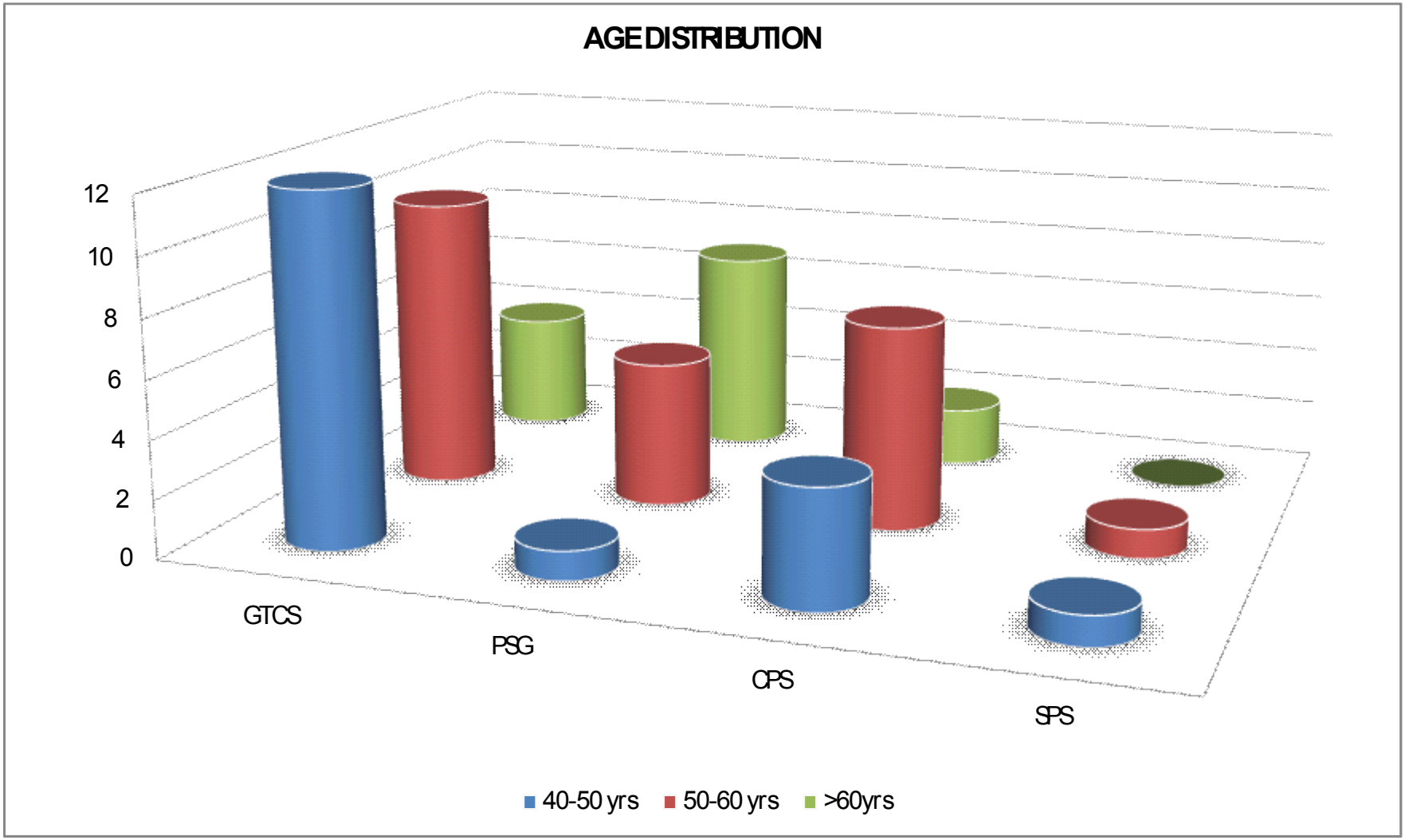

AGE DISTRIBUTION
12
10
8
6
4
2
0
GTCS
PSG
CPS
SPS
40-50 yrs
50-60 yrs
>60yrs

Table.3 SEIZURE TYPE

	Incidence
Generalised tonic clonic	26 (52%)
Simple partial	2 (4%)
Complex partial	9 (18%)
Partial with sec generalisation	13 (26%)

Table.4 PRECIPITATING FACTORS

Precipitating factor	Incidence
Menstruation	2
Emotional upset	9
Sleeplessness	7
Alcohol withdrawal	7
New moon day	5
Fever	4
No factors	16

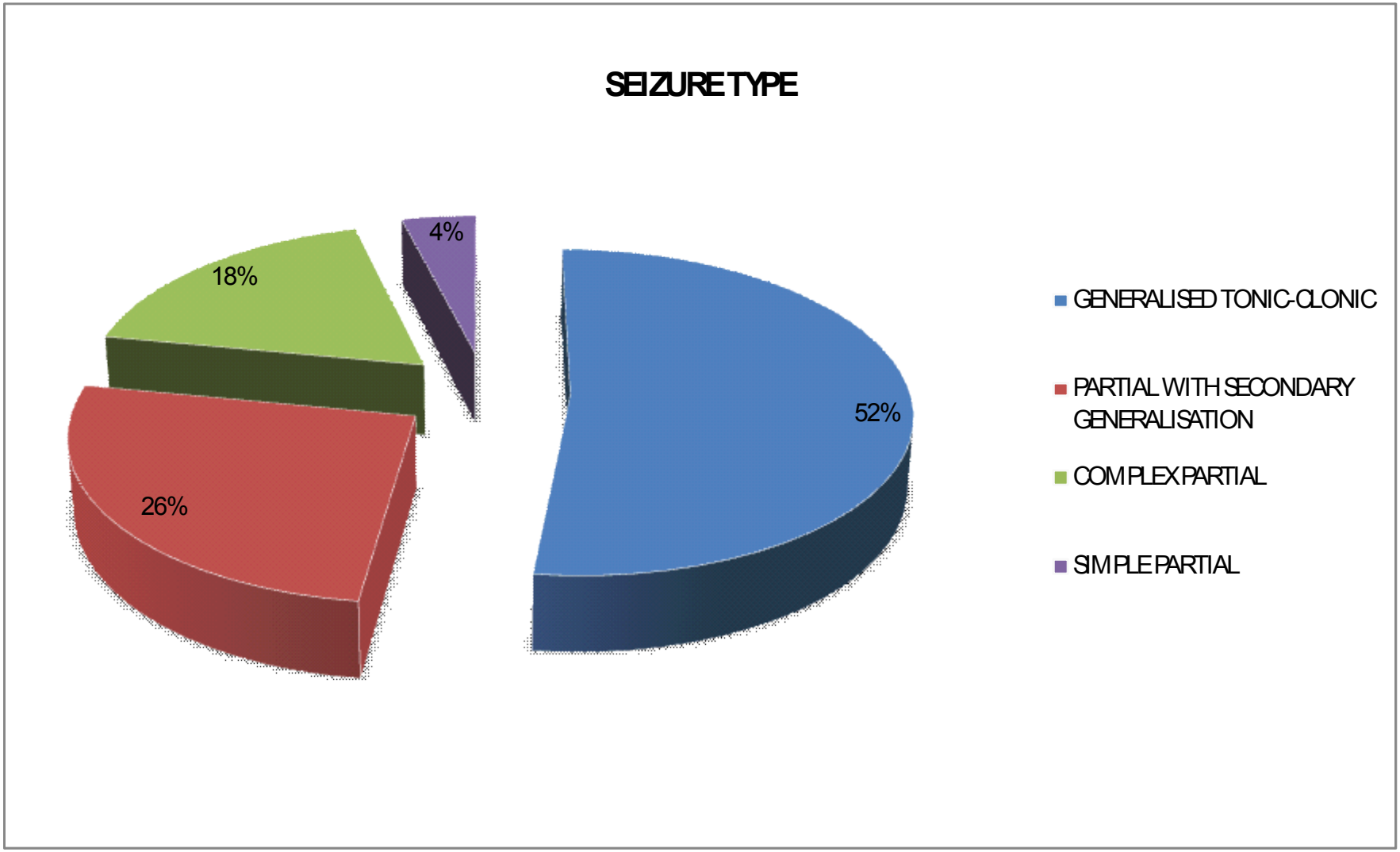

SEIZURE TYPE
4%
18%
52%
26%
GENERALISED TONIC-CLONIC
PARTIAL WITH SECONDARY GENERALISATION
COMPLEX PARTIAL
SIMPLE PARTIAL

Table.5 AURA

Headache	11
Visual*	2
Giddiness/ light headedness	3
Tingling sensation	2
Others**	2

* Visual aura included: micropsia, macropsia, light flashes, dimness of vision, double vision

** Others included: olfactory hallucinations, jamais vu, déjà vu, abnormal sensations/ movements

Table.6 POST ICTAL STATE

Duration	Incidence
<24 hrs	35
>24 hrs	4

Symptoms	Incidence
Headache	22
Asthenia	12
Neurological deficit	4
Automatism	6
Disorientation	19
Excessive sleep	7

Table.7 RELEVANT PAST HISTORY

Head injury	2
Hypertension (inc PIH)	15
Diabetes mellitus	11
Cardiovascular diseases*	8
Respiratory illnesses**	3
Renal transplant	1
Psychiatric	1
Old CVA	10

* Cardiovascular diseases included Rheumatic Heart Disease, Mitral Valve Prolapse, Ischemic Heart Disease

** Respiratory illnesses included COPD, Pulmonary Tuberculosis, Bronchial Asthma

Table.8 FAMILY HISTORY OF SEIZURES

Present	3
Not present	47

Table.9 RELEVANT PERSONAL HISTORY:

Smoking	20
Drugs (Addictions)	5
Alcohol	22
Tobacco	4
Snuff	5

Pork	5

Table.10 EXAMINATION FINDINGS:

Hypertension	21
Neurological deficits	24
Confusional state	19
Meningeal irritation	2
Abnormal fundus	15
Cardiovascular signs	2
Respiratory signs	2
Abdomen signs	1
Others*	2

* Others included: dementia, mental retardation, unconsciousness

NEUROLOGICAL DEFICITS

Hemiparesis	15
Monoparesis	2
Hemi sensory loss	3
Slurring of speech	5
Memory disturbances	1
Dementia	2

FUNDUS CHANGES

Grade I & II hypertensive changes	15

Papilledema	5
Fundal haemorrhages	1

Table.11 BRAIN IMAGING FINDINGS

Intracerebral Haemorrhage	5
Cerebral infarction	12
Subdural haemorrhage	2
Granuloma	4
Diffuse cerebral atrophy	2
Tumours	3
Normal	22

Table.12 EEG FINDINGS

Normal	37
Abnormal	13

EEG ABNORMALITIES

Focal slowing	7
Diffuse slowing	4
Spikes and sharp waves	2

Table.13

CT Scan	EEG	Incidence
Normal	Abnormal	4
Abnormal	Normal	19

Abnormal	Abnormal	9
Normal	Normal	18

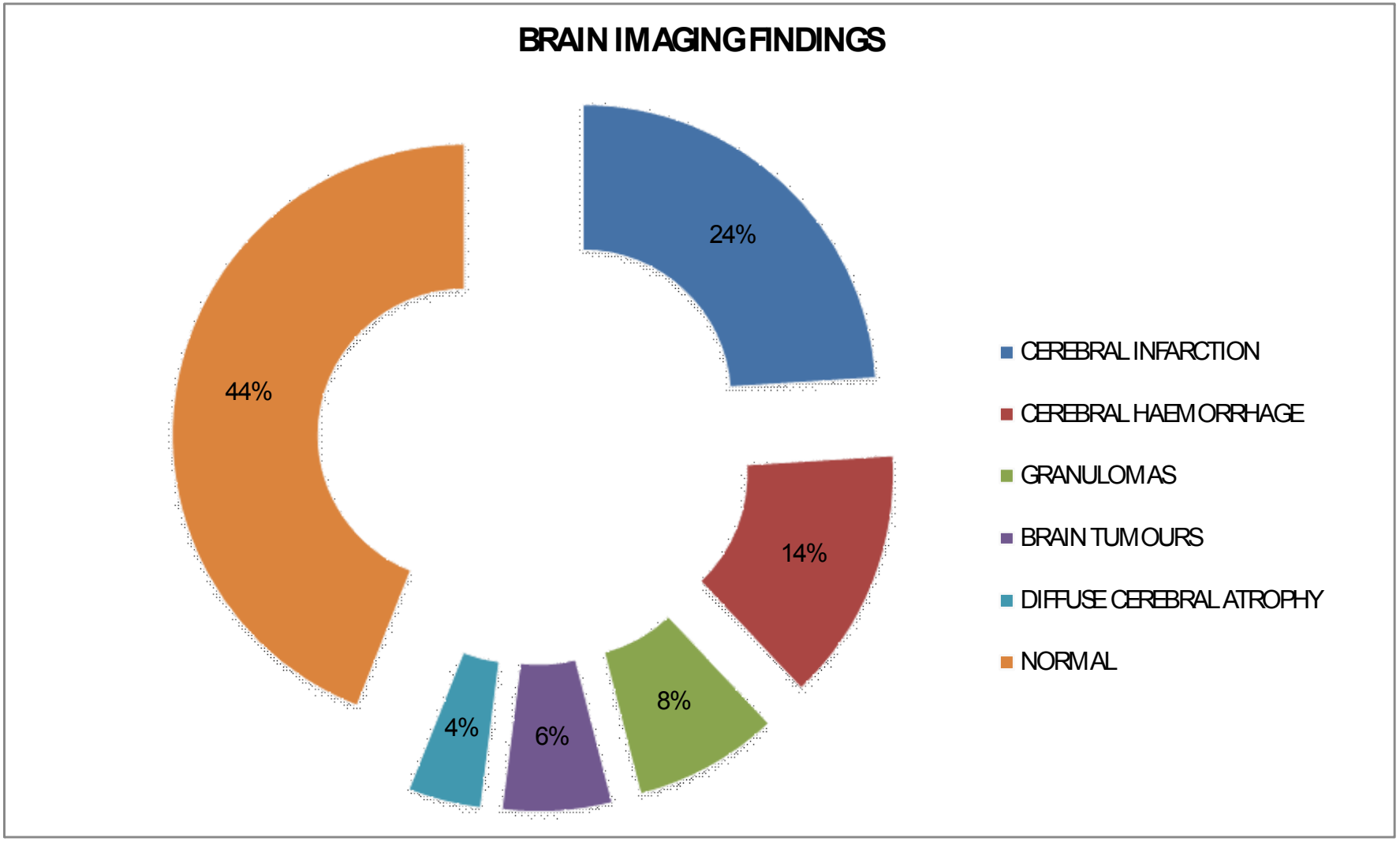

BRAIN IMAGING FINDINGS
24%
14%
8%
6%
4%
44%
CEREBRAL INFARCTION
CEREBRAL HAEMORRHAGE
GRANULOMAS
BRAIN TUMOURS
DIFFUSE CEREBRAL ATROPHY
NORMAL

Table.14

ETIOLOGY	GTCS	CPS	PSG	SPS	TOTAL
Cerebral infarct	2	3	7	-	12
Cerebral haemorrhage/SDH(2)	1	2	4	-	7
Granuloma / Tuberculoma (3) / NCS(1)	-	3	1	-	4
Brain tumour / Secondaries(1) Glioma (1) / Meningioma(1)	-	1	1	1	3
Alcohol withdrawal	6	-	-	-	6
Hypoglycaemia	2	-	-	-	2
Hepatic encephalopathy	1	-	-	-	1
Hyponatremia	1	-	-	-	1
Dementia Diffuse cerebral atrophy	2	-	-	-	2
Cerebral malaria	2	-	-	-	2
Viral encephalitis	1	-	-	-	1
TB- Meningitis	1	-	-	-	1
Hypertensive Encephalopathy	1	-	-	-	1
Unknown	6	-	-	1	7
Total	26	9	13	2	50

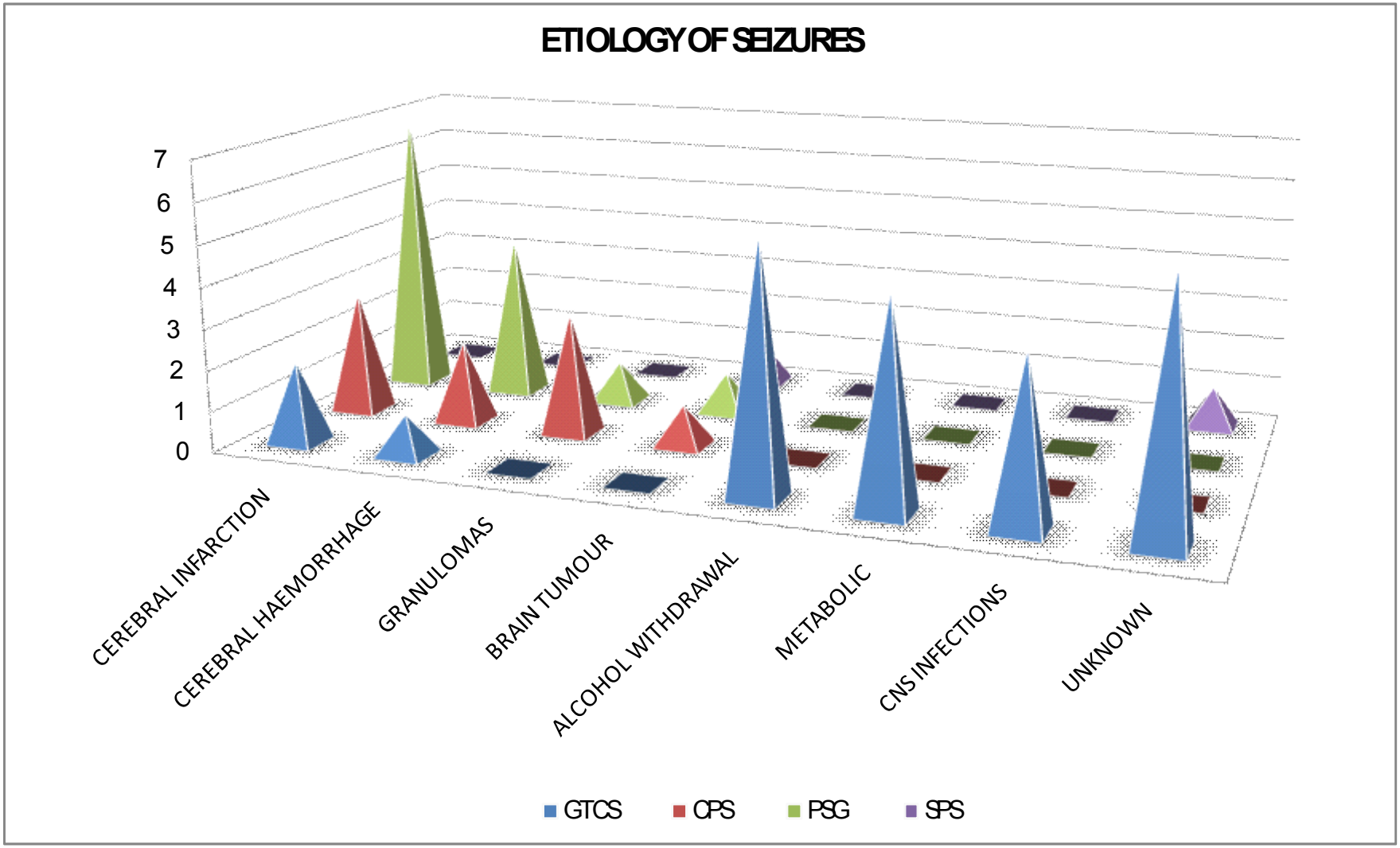
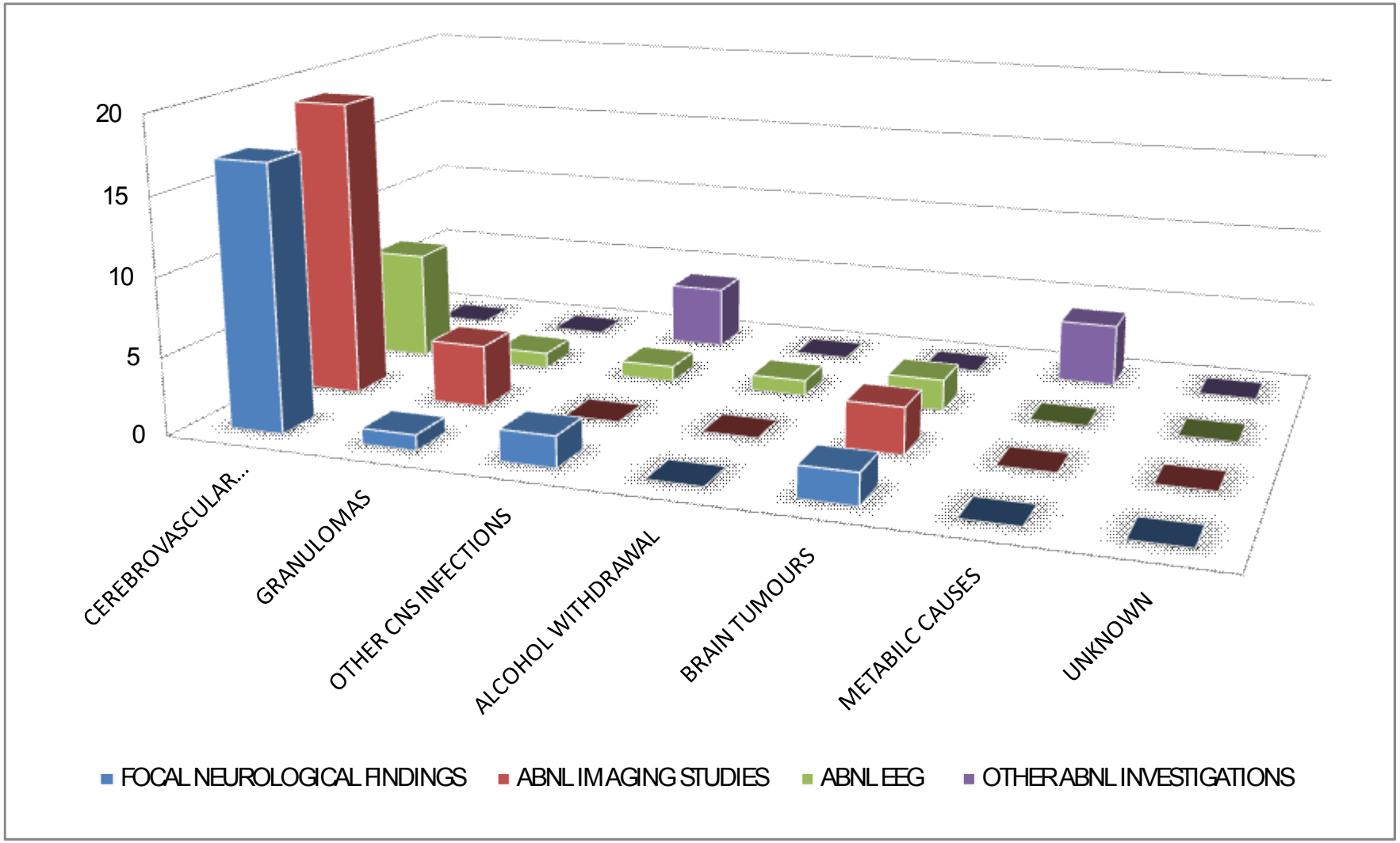

ETIOLOGY OF SEIZURES
7
6
5
4
3
2
1
0
CEREBRAL INFARCTION
CEREBRAL HAEMORRHAGE
GRANULOMAS
BRAIN TUMOUR
ALCOHOL WITHDRAWAL
METABOLIC
CNS INFECTIONS
UNKNOWN
GTCS
CPS
PSG
SPS

Table.15

Etiology	Focal Neurological Findings	Abnl Imaging	ABNL EEG	Other ABNL Investigations
Cerebral infarct(12)	12	12	4	-
Cerebral haemorrhage (7)	5	7	3	-
Granulomas(4)	1	4	1	-
Brain tumour(3)	2	3	2	-
Alcohol withdrawal (6)	-	-	1	-
Hypoglycaemia (2)	-	-	-	2 (Low Blood sugar)
Hepatic encephalopathy (1)	-	-	-	1(abnl LFT)
Hyponatremia (1)	-	-	-	1(low Sr. Na)
Diffuse cerebral atrophy(2)	2 (Dementia)	2	1	-
Cerebral malaria(2)	-	-	-	2(QBC-MP +)
Viral encephalitis(1)	1(neck stiffness)	-	1	1(CSF:HSV IgM +)
TB- Meningitis(1)	1(neck stiffness)	-	-	1 (CSF: ADA+)
Hypertensive Encephalopathy (1)	-	-	-	-
Unknown(7)	-	-	-	-
Total (50)	24(48%)	28 (56%)	13(26%)	8 (16%)


20
15
10
5
0
CEREBROVASCULAR...
GRANULOMAS
OTHER CNS INFECTIONS
ALCOHOL WITHDRAWAL
BRAIN TUMOURS
METABILC CAUSES
UNKNOWN
FOCAL NEUROLOGICAL FINDINGS
ABNL IMAGING STUDIES
ABNL EEG
OTHER ABNL INVESTIGATIONS

DISCUSSION

The goal of evaluation of a patient with paroxysmal episodes of seizures are to (1) determine whether the patient has epilepsy, (2) correctly characterise the type of seizures, (3) identify the potential cause of seizures, (4) determine the course of treatment, if needed, (5) provide patient and the family about the course of disorder[1].

As the age increases, it is more likely the episodes are symptomatic seizures rather than being idiopathic. So when seizures are encountered in adult life, more care should be taken to identify an underlying cause, which could be treatable.

This study is aimed at studying the clinical profile of patients with new onset seizures in adults and finding the etiology with possible investigations.

Following are the inferences derived from the observations made in the study.

AGE:

In this study, though late onset seizures was most common in the age group of 50- 60 yrs (38%), there was not any significant difference

between the other age groups. 36% were seen in the age group 40-50 yrs and 26% in > 70yrs.

de Bittencourt et al.,1996[6],in their study had similar findings. They say that the age-specific incidence in developing countries, is high throughout adulthood, largely related to symptomatic seizures occurring as a result of infection and trauma.

SEX:

Males were 1.6 times more in number than females in patients presenting with late onset seizures. They contributed to 62% of the patients.

The reasons for this might be that male patients have more indirect risk factors like smoking, alcohol, ischemic heart disease etc., Bradley mentions that, *men are 1.0 to 2.4 times more likely to have seizures than women[2]*.

TYPE OF SEIZURES:

Complex partial seizures with or without secondary generalisation have been found to be the most common type of seizures[2].

But in this study, generalised tonic clonic seizures were the commonest (50%), partial seizures with secondary generalisation (26%), complex partial seizures (20%) and the last being simple partial seizures (4%). It might be due to the reason that only inpatients were evaluated in this study. And patients in developing countries bring to notice mostly generalised seizures, ignoring episodes of insignificant seizures like partial seizures without gross disturbances in consciousness.

As the study population comprised only adults, we didn't come across other seizure types like absence or atonic or myoclonic.

CLINICAL FEATURES:

PRECIPITATING FACTORS:

More than 40 factors have been recognised as precipitating factors, of which emotional factors were the commonest (Nakken KD et al, 2005[5]).

In this study, emotional upset (9 patients), sleeplessness (7) and alcohol withdrawal (7) were the most common precipitants.

AURA:

Headache was the most commonest preceding symptom (21 patients). The other common preceding symptoms being giddiness, visual symptoms like light flashes, dimness of vision and tingling sensation.

POST-ICTAL STATE:

Apart from patients with simple partial seizures and complex partial seizures, nearly all the other patients with generalised (GTCS and partial with secondary generalisation) had some form of post ictal symptoms, 4 of them which lasted for more than 24 hours.

Most common symptoms of these being headache (22 patients), disorientation (19), and asthenia(12). Neurological deficits were in the form of hemiparesis in 2 patients, hemi-sensory loss in 1 patient and dysphagia and dysarthria in 1 patient. For these above mentioned patients, neurological deficits were transient and they had no evidence of focal lesions on CT scan.

Those with neurological deficits and structural lesions are discussed later.

PAST HISTORY:

One of the most common etiologies in this study was cerebrovascular accidents. So it can be expected that patients commonly had chronic illness like hypertension (15), diabetes mellitus (11),ischemic heart disease (6). 2 patients had rheumatic heart disease. One with mitral stenosis and second patient with mitral stenosis and aortic regurgitation. Both patients had left atrial clot on echocardiogram and were presumed to have embolic stroke. 2 patients had history of significant head injury in the past. They were found to have chronic subdural hematoma on CT scan. Both patients were alcoholics.

FAMILY HISTORY:

Positive family history was present in just 3 of the patients. Of these 2 patients had cerebrovascular accident with infarct and 1 had undiagnosed cause. So family history does not seem to be relevant in patients with late onset seizures.

EXAMINATION FINDINGS:

21 patients were found to be hypertensive on admission. Of these 8 were known hypertensives with uncontrolled BP. Anti hypertensives

were stepped up for these patients. 10 of the patients had reactive hypertension due to the seizure episode. Their BP came to normal in few days without any medications. And 3 patients were newly diagnosed hypertensives. 1 among them had BP>210/110 mmHg on admission. He had no focal deficits or lesions on CT or any significant inter ictal EEG. He was diagnosed to have seizures due hypertensive encephalopathy. Seizures got controlled with symptomatic treatment and BP control with antihypertensives.

NEUROLOGICAL DEFICITS:

On admission, 24 patients had neurological deficits. Of these 4 had transient deficits with out any focal lesions on imaging and were considered to be due to post ictal state. 2 pateints had features of dementia and were found to have diffuse cortical atrophy on brain imaging later.

Patients with cerebrovascular accidents, acute and chronic had deficits according to the vascular terrtitory affected. The deficits included hemiparesis(15), monoparesis (2), hemisensory loss(3), slurring of speech(5),memory disturbances(1). The rest of the significant examination findings were confusional state (19), meneigeal irritation

(1-cerebral malaria, 1- viral encephalitis). Patients with cardiovascular, respiratory and abdominal pathology had corresponding findings.

BRAIN IMAGING:

28 patients (56%) of patients had significant imaging findings pointing towards the diagnosis. Imaging is one of the important factors determining the etiology of seizures (Table 15). So every patient with new onset of seizures should undergo a CT scan brain. And when required contrast study or an MRI might be needed.

CEREBRAL INFARCTION:

12 patients had cerebral infarcts corresponding to their neurological physical examination.

Of these, 5 seizures occurred within a period of 1 week of onset of stroke (acute symptomatic). 4 of these were partial with secondary generalisation; 1 of these was generalised tonic clonic seizures.

Labovitz L et al[42] state that 5-10% of individuals with cerebrovascular insult experience seizure at the time of onset.

The rest of the 7 patients had seizures after an interval of several months following cerebrovascular accident. Of these, 3 were complex

partial seizures, 1 was generalised tonic clonic seizures, 3 were partial with secondary generalisation.

Adams[4] mentions that patients with infarcts involving cortex, after interval of months develop seizures in about 10 % of patients.

CEREBRAL HAEMORRHAGE:

Of these, 5 were intracerebral and 2 were subdural haemorrhage.

Both the patients with subdural haemorrhage, were alcoholic and gave a remote history of head injury. One of them presented with complex partial seizures and another with partial seizures with secondary generalisation.

GRANULOMAS

TUBERCULOMA:

On CT, tuberculomas were defined as high or low density and round or lobulated masses, which are homogenous and show ring enhancement after contrast administration. They have irregular walls of varying thickness (Tandon et al, 1980). Moderate to marked perilesional edema is frequently associated with parenchymal tuberculomas providing substrate for seizures (Patwari AK et al,

1996[44]).

3 such cases were found in our study, satisfying the CT criteria for tuberculomas. These patients are relatively in younger age group, mostly within 50 years. All were started on anti tuberculous treatment at discharge.

NEUROCYSTICERCOMA:

Garg et al, 2000[45] showed that after contrast administration, majority of neurocysticercomas revealed a ring/ disc enhancing lesion, sometimes with target sign on CT scan.

One of the patients in the study had multiple typical lesion, though there were no subcutaneous nodules. This patient gave history of consuming pork. He was started on treatment with albendazole.

BRAIN TUMOURS:

LeBlanc et al, 1974[34], stated that seizures develop in approximately 35-40% of adults with brain tumours. Upto 60 % of gliomas develop seizures. Though the rate is as low as 20% in case of secondaries.

3 patients in this study, had seizures due to brain tumours. One of

the patients, presented with generalised tonic clonic seizures with a previous history of smoking, COPD and a recent history of hemoptysis. His brain imaging showed mulitiple hypodense metastatic lesions. His chest X Ray showed a mass lesion in the left upper lobe. Tissue diagnosis was later obtained as squamous cell bronghogenic carcinoma.

Another patient with history of surgery for brain tumour 2 months back (proved as glioma) presented with complex partial seizures. One patient who presented with simple partial seizures, had features of meningioma in parasaggital region on MRI brain.

DIFFUSE CORTICAL ATROPHY:

2 patients had symptoms and signs of dementia with their CT scan showing diffuse cerebral atrophy.

Hesdorffer et al, 1996[41] evaluated the risk for seizures associated with dementia and found that dementia increased the risk of seizures by 8- fold.

EEG FINDINGS:

In almost all the patients the EEG was performed at a later date after the seizures had been controlled and patient discharged from the wards. The EEG findings were obtained with a later follow up. So none of the EEGs were taken during the ictal or post-ictal or in the immediate inter-ictal phase.

Of all the patients, only 13 had EEG abnormalities. This comes to around 26% compared to 56% in patients with positive imaging findings which pointed to a specific etiology of seizures.

7 of them showed focal slowing, corresponding to the epileptogenic focus. 4 had diffuse slowing. And 2 had spikes and sharp waves pattern.

Maimoona Siddiqui et al[10] in their study say that Generalized seizures and generalized slowing on EEG were the commonest findings in patients who developed seizures after stroke. The commonest epileptiform discharges were focal sharp and slow waves seen in 9.8% of patients with post stroke seizures.

ETIOLOGY OF NEW ONSET SEIZURES IN LATE

ADULTHOOD.

After complete evaluation, with history, clinical findings, investigations, specifically neuroimaging and electroencephalography, the diagnosis was derived in as high as 43 patients (86%). A diagnosis could not be sought only 14% of the patients.

In similar studies, Agnete mouritzen et al, Ahuja et al[43] had undiagnosed seizures in as high as 38% and 75.1 % respectively. While Henny C et al, Robert MA et al, had seizures of unknown etiology in only 7% and 16% respectively.

The most common etiology was cerebrovascular accidents comprising 38% of the patients. Of this, 24% had occlusive cerebrovascular disease while 14% had haemorrhagic disease. 8(42%) of these patients had seizures within a week after the insult had occured. The rest, 11(58%) patients, had seizures, few months after the cerebrovascular event(late onset).

SR Gupta et al[52] state that 33% of the 90 seizures appeared early (within 2 weeks after the infarction), and 90% of the 30 early seizures appeared within 24 hours after the infarction.

The next common etiology was seizures associated with alcohol. 6 (12%) of the 50 patients had seizures directly related to alcohol. Most of these seizures were related to withdrawal of alcohol. 2 of the patients had seizures after 3 days of abstinence, and were associated with delirium tremens. 3 had seizures within 48 hours of abstinence. 1of them had seizures during acute alcohol intoxication. Apart from these, alcohol has been indirectly involved in few other patients; 2 patients with sub dural hematoma, 1 patient with alcoholic liver disease with hepatic encephalopathy, 1 patient with hypoglycaemic seizures in whom alcohol and oral hypoglycemics seem to have precipitated the attack.

Granulomas and tumours were one of the important causes of partial seizures. They constituted around 8% and 6% of the patients respectively.

Other CNS infections were seen in 4 of the patients. Of these, 2 had cerebral malaria, 1 herpes encephalitis and 1 tuberculous meningitis. The last patient tested positive for retroviral infection also.

2 of the patients, both above 60years with generalised seizures and features of dementia had diffuse cerebral atrophy.

The rest of them were generalised seizures and had underlying

systemic causes. These are hypoglycaemia (2), hyponatremia(1), hepatic encephalopathy(1) and hypertensive encephalopathy(1).

7 patients had undiagnosed cause for seizures in spite of meticulous examination, investigations, imaging and EEG.

In most of the studies, trauma has been mentioned as an important cause of seizure. But in this study, except for the 2 patients with subdural haemorrhage, no other seizures were associated with trauma. This might be due to the reason that all the patients were chosen from medical wards and trauma wards were not taken into the study.

From the study, it is clear that most of the seizures in late adulthood are symptomatic (86%).

14 of them were remote symptomatic, while the rest (26) had acute symptomatic seizures. So every care must be taken to evaluate the cause of new onset seizures in adults.

CONCLUSIONS

1. Though late onset seizures was most common in the age group of 50-60 yrs (38%), there was not any significant difference between the other age groups. 36% were seen in the age group 40-5o yrs and 26% in > 70yrs.

2. Males were 1.6 times more in number than females in patients presenting with late onset seizures.

3. Generalised tonic clonic seizures were the commonest (50%), then partial seizures with secondary generalisation (26%), complex partial seizures (20%) and the last being simple partial seizures (4%).

4. Family history does not seem to be relevant in patients with late onset seizures.

5. 28 patients (56%) of patients had significant imaging findings pointing towards the diagnosis. Imaging is the most important factors determining the etiology of seizures. So every patient with new onset of seizures should undergo some form of imaging, CT- Plain/ contrast or MRI- Brain depending on the situation as a part of evaluation.

6. EEG did not contribute much to the diagnosis of etiology of the seizures.

7. The most common etiology for late onset seizures was cerebrovascular accidents, ischemic events more commonly causing seizures than haemorrhagic events.

8. This was followed by alcoholic withdrawal state, then granulomas and brain tumours.

9. From the study, it is clear that most of the seizures in late adulthood are symptomatic (86%). So every care must be taken to evaluate the cause of new onset seizures in adults.

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PROFORMA

Name:
Age: Sex:
Address:
DOA: DOD:
IP.No:
Occupation:
Presenting complaints:

Details about seizures:

- First attack:
- Last attack:
- Type of seizures:
- Part of body involved:
- Duration:
- Frequency:
- Maximum period of freedom:
- Time of attack:
- Precipitating factors:
- Aura:
- Post-ictal symptoms:
- Automatisms

Past h/o:

- Hypertension, inc. PIH
- Diabetes mellitius
- Head injury
- Previous stroke
- Cardiovascular (RHD/ MVP/ IHD)
- Respiratory illnesses(COPD/ PTB/ BA)
- Renal transplant
- Psychiatric illnesses
- Jaundice

Family h/o:
Personal h/o:

- Smoking
- Drugs
- Alcohol
- Snuff

- Other addictions
- Pork
- Sleep
- Sexual promiscuity

On examination:
General examination:
BP: PR: RR: Temp:
Neurocutaneous markers:

Central nervous system:
- Focal deficits:
- Confusional state:
- Fundus:

Cardiovascular system:
Respiratory system:
Abdomen:

Investigations:
Urine routine:
Complete blood count:
Blood sugar:
Blood urea: Sr. Creat: Sr. Electrolytes:
VDRL: HIV- ELISA: QBC-MP:
Sr. Cholesterol:
ECG: Chest X-Ray:
X-Ray Skull:
CSF Analysis:
CT Scan- Brain:
EEG:

MASTER CHART

S. No.	AGE/ SEX	SIGNIFICANT HISTORY	SEIZURE/ PPT/AU/PIC	SIGNIFICANT SIGNS	SIGNIFICANT INVESTGATIONS	IMAGING	EEG	ETIOLOGY OF SEIZURE
1.	68/M	SHT/IHD/OLD CVA(8m)/	GTCS/ SL/AU/PIC	RT. RESIDUAL HEMIPARESIS		ATROPHIC CHANGES/ CHRONIC INFARCT IN LT PARIETAL REGION	N	POST STROKE (ISCHEMIC)
2	51/M	RHD-MS/AR/	GTCS/ -/-/PIC	SHT/ ALTERED CONSCIOUSNESS/ LT.HEMIPARESIS/ CVS SIGNS OF MS/AR/PHT	ECHO- RHD-MS/AR/LA CLOT	RT ACUTE INFARCT IN CAPSULOGANGLIONIC REGION/ SURROUNDING EDEMA	A	ACUTE EMBOLIC STROKE
3.	58/M	SHT/DM	GTCS/ -/-/PIC	SHT/ SEMICONSCIOUS/ RT HEMIPARESIS/ PAPILLEDEMA		ACUTE HE- LT CAPSULOGANGLIONIC/ MIDLINE SHIFT	N	ACUTE HAEMORRHAGIC STROKE
4.	43/M	SM/ALC/ PORK	CPS/ EM/-/-	NO FOCAL DEFICITS		MULTIPLE RING ENHANCED LESIONS	N	NEURO CYSTICERCOSIS
5.	52/F	RHD-MS/ OLD CVA(6m)	PSG EM/AU/PIC	RT RESIDUAL HEMIPARESIS/ CVS SIGNS OF MS	ECHO- RHD-MS/ LA CLOT	CHRONIC INFARCT LT FRONTO PARIETAL CORTEX	A	POST STROKE (EMBOLIC)
6	58/F	SHT/DM/IHD	PSG/ -/-/PIC	SHT/ RT HEMIPLEGIA/ MOTOR APHASIA		ACUTE INFARCT- RT PARIETO OCCIPITAL REGION	N	ACUTE ISCHEMIC STROKE
7.	73/F	SHT/ OLD CVA (16m) SN/PORK	CPS/ NM/AU/-	RESIDUAL LT U/L MONOPARESIS		CHRONIC INFARCT RT. PARIETAL CORTEX	A	POST STROKE (ISCHEMIC)
8.	44/M	SHT/ ALC	GTCS/ ALC/ -/PIC	ALTERED CONSCIOUSNESS/ TREMORS		N	N	ALCOHOL WITHDRAWAL
9.	43/M	SM/ALC/ DR	GTCS/ ALC/-/PIC	ALTERED CONSCIOUSNESS/ TREMORS/ NO FOCAL DEFICITS		N	A	ALCOHOL WITHDRAWAL

S. No.	AGE/ SEX	SIGNIFICANT HISTORY	SEIZURE/ PPT/AU/PIC	SIGNIFICANT SIGNS	SIGNIFICANT INVESTGATIONS	IMAGING	EEG	ETIOLOGY OF SEIZURE
10.	56/M	SM/ALC	GTCS FE/-/PIC	NO FOCAL DEFICITS		N	N	UNKNOWN
11.	41/M	SM/ALC	GTCS -/-/PIC	ALTERED SENSORIUM/ NO FOCAL DEFICITS	QBC-MP +	N	N	CEREBRAL MALARIA
12.	66/M	COPD SM/ALC/SN	PSG/ SL/AU/PIC	SHT/ NO FOCAL DEFICITS	CXR- MASS IN LT. UPPER LOBE. Bx- SQUAMOUS CELL CA	MULTIPLE HYPODENSE LESIONS RT FRONTO PARIETAL REGION	N	BRONCHOGENIC CA WITH SECONDARIES
13.	68/F	DM/IHD	PSG/ -/-/PIC	SHT/ RT HEMIPARESIS/ RT HEMISENSORY LOSS/ SLURRED SPEECH/ PAILLEDEMA		ACUTE HGE CAPSULOGANGLIONIC REGION	A	ACUTE HAEMORRHAGIC STROKE
14.	46/F	OLD PTB- DEFAULTER	CPS/ SL/AU/-	NO FOCAL SIGNS	CXR- RT APICAL FIBRO CAVITY	RT FRONTAL TUBERCULOMA	N	TUBERCULOMA
15.	56/M	SHT/ ALC/SM/ PORK	PSG/ -/-/PIC	SHT/ LT HEMIPARESIS/ LT HEMISENSORY LOSS		ACUTE INFARCT IN RT PARIETO OCCIPITAL REGION	N	ACUTE ISCHEMIC STROKE
16.	53/M	SHT/ ALC/SM/ DR	PSG/ EM/-/PIC	SHT/ ALTERED SENSORIUM		RT SUB DURAL HE COMPRESSING PARIETO OCCIPITAL REGION	A	SUBDURAL HAEMORRHAGE
17.	41/F		PSG/ ME/AU/PIC		Mx +	RT FRONTO PARIETAL TUBERCULOMA WITH PERILESIONAL EDEMA	N	TUBERCULOMA
18.	56/F	DM	GTCS/ -/-/PIC	NO FOCAL DEFICITS	RBS<30 MG/DL	N	N	HYPOGLYCEMIA
19.	72/M	SHT/ ALC	GTCS/ SL/-/PIC	SHT/ DEMENTIA		DIFFUSE CORTICAL ATROPHY	N	DEMENTIA

S. No.	AGE/ SEX	SIGNIFICANT HISTORY	SEIZURE/ PPT/AU/PIC	SIGNIFICANT SIGNS	SIGNIFICANT INVESTGATIONS	IMAGING	EEG	ETIOLOGY OF SEIZURE
20.	68/M	SHT/ BRAIN TUMOUR-GLIOMA (POST OP-2m) SM/ALC	CPS/ SL/AU/-	SHT/ ALTERED SENSORIUM/ DYSPHASIA/ PAPILLEDEMA		RECURRENT MASS IN PERIETO OCCIPITAL REGION	N	GLIOMA- POST OPERATIVE, RECURRENT TUMOUR
21.	44/M	ALC	GTCS/ -/-/PIC	SHT/ ALTERED SENSORIUM/ NECK STIFFNESS/ PAPILLEDEMA	CSF- ADA+ PRO↑, SUG↓, HIV-ELISA +	N	N	TB- MENINGITIS
22.	54/M	ALC/SM	GTCS/ NM/-/PIC	N		N	N	UNKNOWN
23.	53/M	SM	GTCS EM/-/PIC	BP>210/110, ALTERED SENSORIUM/ NO FOCAL DEFICITS/ PAPILLEDEMA		N	N	HYPERTENSIVE ENCEPHALOPATHY
24.	59/F	DM/ OLD CVA (8m)	CPS/ FE/ -/PIC	RESIDUAL RT MONOPARESIS		LT FRONTO PARIETAL CHRONIC INFARCT	N	POST STROKE (ISCHEMIC)
25.	54/F	SHT/ OLD CVA (7m)	CPS/ EM/AU/-	SHT/ RESIDUAL LT HEMIPARESIS		CHRONIC HGE- RT FRONTO PARIETAL REGION	A	POST STROKE (HAEMORRHAGIC)
26.	70/M	ALC	GTCS/ EM/-/PIC	NO FOCAL DEFICITS		N	N	UNKNOWN
27.	43/F		GTCS/ -/-/PIC	NO FOCAL DEFICITS	QBC-MP +	N	N	CEREBRAL MALARIA
28.	46/M	ALC/SM	GTCS/ ALC/-/PIC	NO FOCAL DEFICITS		N	N	ALCOHOL WITHDARWAL
29.	42/M	SM	CPS/ EM/-/-	NO FOCAL DEFICITS	Mx +	LT FRONTO PARIETAL TUBERCULOMA WITH PERILESIONAL EDEMA	N	TUBERCULOMA
30.	52/M	SHT/ ALC	GTCS/ NM/-/PIC	NO FOCAL DEFICITS		N	N	UNKNOWN

S. No.	AGE/ SEX	SIGNIFICANT HISTORY	SEIZURE/ PPT/AU/PIC	SIGNIFICANT SIGNS	SIGNIFICANT INVESTGATIONS	IMAGING	EEG	ETIOLOGY OF SEIZURE
31.	56/M	ALC/SM	PSG/ NM/AU/PIC	ALTERED SENSORIUM		LT CHRONIC SUBDURAL HEMATOMA COMPRESSING FRONTO PARIETAL REGION	N	SUBDURAL HEMATOMA
32.	52/F	SHT/CKD	GTCS/ AGE/-/PIC	ALTERED SENSORIUM/ NO FOCAL DEFICITS	SR.Na- 106 MG/DL	N	N	HYPONATREMIA
33.	74/F		GTCS/ EM/-/PIC	DEMENTIA		DIFFUSE CERBRAL ATROPHY	N	DEMENTIA
34.	56/F	DM	SPS/ SL/AU/-	NO FOCAL DEFICITS		MENINGIOMA- LT PARASAGGITAL REGION	N	TUMOUR-MENEIGIOMA
35.	47/F	SN	SPS/ -/AU/-	NO FOCAL DEFICITS		N	N	UNKNOWN
36.	45/F		GTCS/ FE/-/PIC	SHT/ ALTERED SENSORIUM/ NECK STIFFNESS/ FUNDAL HAEMORRHAGES	CSF- LYMPHOCYTIC PLEOCYTOSIS/ HSV IgM +	MRI- HYPODENSITIES IN LT TEMPORAL REGION	A	VIRAL ENCEPHALITIS
37.	62/M	OLD CVA (8m)/ /SM/ PORK	PSG/ EM/AU/PIC	RESIDUAL RT HEMIPARESIS/ SLURRED SPEECH/ MEMORY DISTURBANCES		CHRONIC INFARCT IN LT PARIETAL CORTEX AND CORONA RADIATA	A	POST STROKE (ISCHEMIC)
38.	48/F		GTCS/ ME/-/PIC	N		N	N	UNKNOWN
39.	51/M	SHT/IHD/ OLD CVA(6m) ALC	CPS/ SL/AU/-	LT RESIDUAL HEMIPARESIS		CHRONIC HGE IN RT PARIETAL REGION	A	POST STROKE (HAEMORRHAGIC)
40.	48/M	DCLD/ PHT/ GR III HEPATIC ENCEPHALOPATHY ALC/DR	GTCS/ -/-/PIC	ASTERIXIS/ NO FOCAL DEFICITS/ ICTERUS/ ABD-ASCITES/ SPLENOMEGALY	ABNORMAL LFT/ USG- CIRRHOSIS WITH PHT	N	A	HEPATIC ENCEPHALOPATHY
41.	49/M	SHT/DM	GTCS	NO FOCAL DEFICITS	RBS<40 MG/DL	N	N	HYPOGLYCEMIA

S. No.	AGE/ SEX	SIGNIFICANT HISTORY	SEIZURE/ PPT/AU/PIC	SIGNIFICANT SIGNS	SIGNIFICANT INVESTGATIONS	IMAGING	EEG	ETIOLOGY OF SEIZURE
		SM/ALC	ALC/-/PIC					
42.	59/F	SHT	GTCS/ SL/AU/PIC	NO FOCAL DEFICITS		N	N	UNKNOWN
43.	42/M	ALC/ DR	GTCS/ ALC/-/PIC	ALTERED SENSORIUM/ TREMORS		N	N	ALCOHOL WITHDRAWAL
44.	69/M	SM	PSG/ -/-/PIC	SHT/ GLOBAL APHASIA/ RT HEMIPARESIS/ RT HEMISENSORY LOSS/ SLURRED SPEECH		ACUTE INFARCT RT PARIETO TEMPORAL	N	ACUTE ISCHEMIC STROKE
45.	64/M	SHT/DM/IHD/OLD CVA(4m)/ SM/ PORK	PSG/ FE/AU/PIC	SHT/ RESIDUAL RT HEMIPARESIS		CHRONIC INFARCT LT CAPSULO GANGLIONIC REGION	A	POST STROKE (ISCHEMIC)
46.	42/M	ALC/SM/DR/SN	GTCS ALC/-/PIC	TREMORS/ ALTERED SENSORIUM		N	N	ALCOHOL WITHDRAWAL
47.	62/M	OLD CVA(18 m)/ SM	PSG/ -/-/PIC	SHT/ LT RESIDUAL HEMIPARESIS/ SLURRED SPEECH		CHRONIC HGE IN RT PARIETAL REGION	A	POST STROKE (HAEMORRHAGIC)
48	72/M	SHT/DM	PSG/ -/-/PIC	SHT/ ALTERED SENSORIUM/ LT HEMIPARESIS/ SLURRED SPEECH		ACUTE INFARCT IN PARIETO TEMPORAL CORTEX WITH SURROUNDING EDEMA	N	ACUTE ISCHEMIC STROKE
49.	53/M	OLD CVA (2y) SM	CPS/ NM/AU/-	SHT/ RESIDUAL RT HEMIPARESIS		CHRONIC INFARCT IN LT PARIETAL CORTEX AND CORONA RADIATA	N	POST STROKE (ISCHEMIC)
50.	42/M	ALC/ SM/SN	GTCS/ ALC/-/PIC	TREMORS/ ALTERED SENSORIUM		N	N	ALCOHOL WITHDRAWAL

ABBREVIATIONS FOR MASTER CHART

GTCS	-	Generalised tonic - clonic seizures
CPS	-	Complex partial seizure
SPS	-	Simple partial seizures
PSG	-	Partial seizures with secondary generalization
PPT	-	precipitating factors
EM	-	emotional upset
SL	-	sleeplessness
ALC	-	alcohol
NM	-	new moon day
FE	-	fever
AU	-	aura

PIC	-	post ictal confusion
SHT	-	systemic hypertension
DM	-	diabetes mellitus
COPD	-	chronic obstructive pulmonary disease
IHD	-	ischemic heart disease
PTB	-	pulmonary tuberculosis
SM	-	smoking
SN	-	snuff
DR	-	drugs/ addictions